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COMMON VASCULAR PROBLEMS OF THE LOWER EXTREMITIES

PART I *Considerations in Vascular Insufficient States of the Lower Extremities:*

The Anatomic, Physiologic and Pathologic

JOSEPH BENINSON, M.D.*

Modern day medical care has extended the life span of man and concomitantly, the incidence of degenerative disease. Not least in this progression is the ever increasing number of people who now live to acquire vascular insufficiency. Dr. Gunnar Bauer of the Marie Stadt General Hospital in Sweden, in discussing this problem at the Fourteenth Congress of the International Society of Surgery in Paris in 1951, estimated that one person in every five could expect to suffer from improper venous return from the lower extremities in one form or another and noted, "that with the exception of rheumatism, this condition (specially post-thrombophlebitic sequelae) heads all other illnesses causing invalidity."¹

ANATOMICAL CONSIDERATIONS

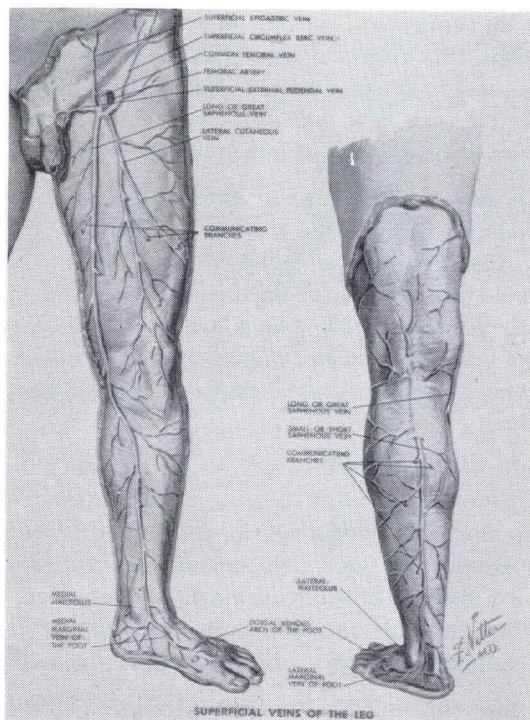
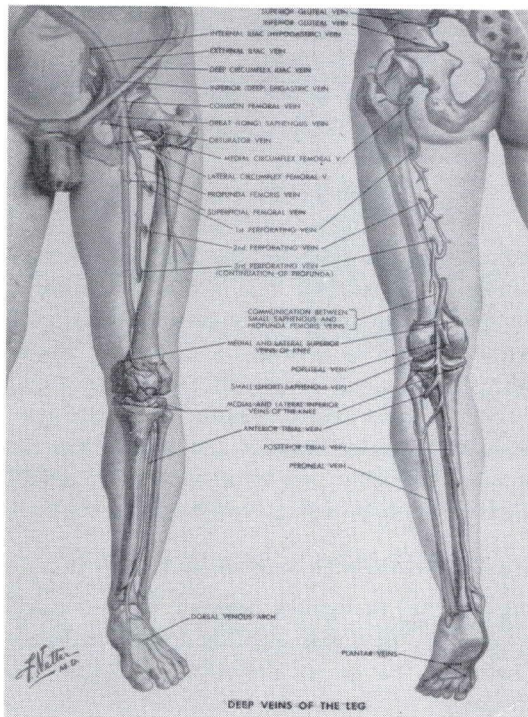
Before embarking on the interaction between the structural, physiological and pathological forces involved in vascular stasis of the lower extremities, some fundamental knowledge must be reviewed. To do so, the author will draw freely on Burch's "A Primer of Venous Pressure."²

A review of the gross anatomy involved is afforded by Figures 1 and 2³ which show the superficial and deep veins of the lower extremities and their relationship to the adjacent structures. It is evident that whereas the deep veins have the support of the muscle masses with their connective tissue envelopes and an outer connective tissue sleeve of considerable thickness, the superficial vessels, in the main, lie external to that outer sleeve having only the fat, subcutaneous tissue and skin for support.

The venous valves are almost without exception bicuspid in the lower extremities. These are spaced at intervals in both the deep and superficial veins as well as in the communicating veins. They are also to be found at the point where a vein emerges from a muscle just prior to the point at which it empties into a collecting vein; however, the intramuscular portion of this vein is avalvular. This mechanism constitutes the "peripheral heart" pumping station so that normally muscular contraction can spew blood under increased pressure into the venous circulation and speed the return to the heart.

Essentially, these valves are reflections of vein intima which develop early in fetal life in large numbers; however, many of these never reach full development and their number decreases as the fetus grows. After the infant is born the number of valves decreases with age so that when needed most they are present least. Actually, to date, no one has made an accurate study of the number of valves present in different age groups. Furthermore, Edwards and Edwards⁴, and many other investigators have shown that following complete thrombosis of any vein of the lower extremities, there

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Vascular Problems of the Lower Extremities

eventuates an anatomically and functionally valveless, rigid venous segment or entire vein with an irregular lumen. The valves of communicating veins or muscular veins are sometimes affected by this process simultaneously.

Histologically, the veins tend to be thin walled and wide lumined compared to the adjacent arteries. The thinner walls are the result of relatively small amounts of smooth muscle, elastic tissue and collagenous fibers being present, for veins consist largely of connective tissue. These three layers of intima, media and adventitia are preserved until the level of the venules and capillaries. It is perhaps significant that veins have a richer supply of vasa vasora than arteries, even down to those of 1.0 mm. diameter. Perhaps lowered oxygen tension and slowed flow dictate this richer supply and it would be of interest to know what part, if any, this blood supply plays in keeping the thrombosed vessels viable until they can recanalize.

Because of their thin walls, veins are pliable and tend to collapse almost completely when emptied of blood unless they are situated where connective tissue trabeculae act as guy-wires. These trabeculae tend to counteract the effect of rising tissue pressures proportionately. This is the case in the extremities.

The internal saphenous vein is wider distally than at its proximal terminus to allow for the inflow from the communicating veins which are most plentiful distally.⁵ The communicating veins enter the deep veins at an oblique angle (toward the heart) and it is the incompetency of these communicating vein valves that Muir, Mucklow and Rains have demonstrated to be present in their study of varicosities by venography.

The lymphatics of the lower extremities are found in the dermis—in the superficial and deep plexuses particularly of the plantar skin of the foot—as well as along the superficial and deep vessels, in joint capsules, periosteum, bone and muscle tendons.⁷ Their presence in muscles is disputed.

Most investigators believe that lymphatics begin as offshoots of blood vessels which soon lose their connections and then form capillary tubes which anastomose freely and have many cul-de-sacs. The capillaries themselves are avalvular but form collecting vessels which are richly supplied with bi- or tri-cuspid valves and these drain through regional lymph nodes and/or an occasional intercalated node. In both types of nodes the capillaries break up into wide-lumened capillary sinuses which again reform to produce collecting vessels on their way to the cisterna chyli, thoracic duct and the juncture of the left jugular and left subclavian veins.

Histologically, one finds the three layers as noted for the veins but lymphatic vessels are thinner, the layers less apparent, and in the normal lower extremities the collecting vessels are rarely more than 0.5-0.75 mm. in diameter. Anastomoses between collecting vessels are more frequent than they are in their companion veins.

It is believed that whereas the veins have marked regenerate powers, the lymphatics heal and regenerate slowly and that lymph nodes probably do not regenerate unless under pathologic stimulus.

A. J. Reyniers⁸ and his group have clearly demonstrated the intriguing fact that the development of lymphoid tissue is definitely affected by the presence or absence of infection in the environment of the individual.

In man, it has been estimated that between 1½ and 3 liters of lymph flow through the thoracic duct in 24 hours. The majority of this comes from the liver and intestines. During rest the flow from the lower extremities is believed to be extremely small but it is increased by activity and massage. The rate of flow in pathologic states associated with vascular insufficiency of the lower extremities is not known.

Crissman and Blalock⁹ report that pulsations transmitted from arteries to adjacent lymphatics also aid the return flow of lymph.

McMaster and Hudach¹⁰ using vital blue dyes and following the streamers that develop in the lymphatics find that when the limb hangs down after injection, none or almost none of the dye streams heartward. If the subject be supine and at rest, then short, pale streamers develop slowly. Following massage, elevation of the leg or exercise they develop into rapidly moving, long, deep colored, lymphatic streamers. Following the release of venous obstruction with its reactive hyperemia, which latter is also seen following violent exercise, the largest, darkest and most rapidly moving streamers develop.

There are no streamers in patients with idiopathic lymphedema or in patients with cardiac decompensation. The latter apparently also have venous valvular decompensation probably caused by overdilatation of the veins, for venous valves are said to become functionally patent if their diameters increase more than 40%. In cardiacs, the streamers can be made to go distally, by massage, which supports this possibility of valvular failure for this could not be accomplished in normal subjects. Similarly, with the onset of diuresis following nephrosis, marked streamer formation ensues. Further, McMaster and Hudach¹⁰ show that every intradermal injection is in part a lymphatic injection and that the transport of foreign substances by lymphatics is more rapid than the volume of lymph flow indicates, for normally the lymphatics are flat and a little fluid moves a long way.

The exact role of the lymphatics in the lower extremities has not been detailed entirely; however, the work of McMaster,¹⁰ Moyer and Butcher,¹¹ Kinmonth, Taylor and Harper¹² and others have clarified some of the thinking on this subject.

McMaster¹³ points out that the formation of lymph takes place in the main through the walls of lymphatic capillaries and that the larger collecting channels act chiefly, if not entirely, as conveyors of this fluid to the blood. He as well as Field, Drinker and White¹⁴ confirm the hypothesis of Gaskell and also of Starling that the intralymphatic capillary pressure is lower than the tissue pressure. This does not bring about collapse but rather expansion of the lymphatic capillaries for as tissue pressure mounts, the capillary walls — like most veins — have guy-wire-like fibrillar connections to the adjacent connective tissue elements and the vessel walls are pulled apart.

Lymph stasis as exemplified by phlegmasia alba dolens is caused by primary lymphatic obstruction. The veins have naught to do with the initial accumulation of lymphatic tissue fluid. Homans¹⁵ and Homans and Zollinger¹⁶ have demonstrated experimentally that ligation of the veins does not prevent the phlegmasic swelling from disappearing once the lymphatic obstruction is relieved.

PHYSIOLOGIC CONSIDERATIONS

Under physiologic conditions then, we have a closed system including the heart, arterio-venous vasculature and lymphatics which tend to follow the fluid principles as

Vascular Problems of the Lower Extremities

set out by Poisseuille's and Bernoulli's laws. (For a simple explanation of these concepts see Burch's Primer²). Practically speaking, these laws explain why the venous pressure exerted is greater in smaller (peripheral) veins yet the force supported per unit length of these smaller veins is less than that on the veins close to the heart which have greater cross-sectional diameter (lateral pressure is proportional to surface area).

Eyster¹⁷ gives a graphic representation of the hydrostatic pressures produced by standing, when he translates height into pressure, saying that for each vertical cm. in height the effect of gravity is equal to about 10 mm. of water or about 0.74 mm. of mercury.

This then makes for the physiologic, hydrostatic, pressure-gradient which is normally present in man.

The general factors affecting the normal return of blood from the tissues to the heart are:

1. Contraction of cardiac muscle
2. Pressure originating in the heart (*vis a tergo*)
3. Negative intrathoracic pressure
4. Relative volume of blood in arterial and venous systems
5. Venous tone
6. Gravity
7. Tissue pressure

The mean venous pressure in the lower extremity of a normal, erect, adult male varies from about 1400.0 mm. of water in the dorsalis pedis vein to about 1000.0 mm. of water in the upper end of the great saphenous vein.² Knowing about the phlebostatic level — that point (about the third interspace, at the sternum) where the venae cavae join and enter the right atrium under 0.0 mm. pressure — then makes it clear that whereas the normal, resting, supine individual has a mean venous pressure of 180.0 mm. of water at the ankle, the effect of standing is equivalent to adding thereto the hydrostatic pressure present in the length of a column of fluid from the dorsum of the foot to the phlebostatic level of the heart. In the physiologic state the weight of this column of fluid is distributed between the consecutive valves which divide the vein into pressure-segments².

A review of the literature reveals the following information regarding values under varying conditions in the normal veins of the lower extremities.

Pollack and Wood¹⁸ report the direct, mean, venous pressures in the saphenous vein at the ankle (converted from mm. of mercury to mm. of water) in normal man to be as follows:

| | |
|--------------------------|---------------------|
| Recumbent position | 158.0 mm. of water |
| Sitting position | 756.0 mm. of water |
| Quiet, standing position | 1172.0 mm. of water |

This pressure shows an average drop of about 810. mm. of water with walking, and a return to normal in about 30 seconds after walking ceases. Thirty seconds is the time required for the blood capillaries to refill the veins when the valves are competent. This is the basis of the tests devised for venous incompetency.

This correlates reasonably well with Carrier and Rehberg's¹⁹ report that the normal, average, direct, venous pressure (converted to mm. of water) in the dorsal vein of a relaxed foot hanging over the edge of a chair on which the patient is standing with the other foot is as follows:

| | |
|----------------|--------------------|
| 0 time | 860. mm. of water |
| 5 minutes | 970. mm. of water |
| 6 minutes | 1020. mm. of water |
| over 6 minutes | no change |

During a 15 minute time interval the fluid loss into the tissues in this experiment distends the dependent foot from an initial circumference of 27.0 to 29.0 cm.

These volume figures correlate with the finding of Youman et al²⁰ that standing at a 75° angle for one hour causes the volume of the leg of normal subjects to increase over 25% without any pitting edema. This is accompanied by an increase of 18 — 40% in the serum protein concentration and an increase of 29 — 65% in the colloid osmotic pressure in the leg veins. From these figures they calculated that the increase in tissue pressure is three to five times as important in limiting fluid loss from the blood as is the increase in colloid osmotic pressure.

McMaster²¹ found that in the slow edema associated with irritant chemicals or topical applications, the rise in the interstitial pressure of skin was slight. Also, he determined that the interstitial resistance of dermis in man could be measured directly by the edema fluid in the usual edematous skin and that it was about 5.0 mm. of water higher than interstitial pressure. In the boggy, edematous skin no measurement could be made, for although it appeared to feel like ordinary edema no free fluid was present.

Landis²² listed the factors favoring edema formation and correlated them with clinical examples.

Montgomery and Zintel²³, in a recent compilation of the direct venous pressures just above the ankle in normal subjects with primary varices and in post-thrombophlebics, noted that marked pressure differences were apparent only when the patient was walking.

In still another report, Wells et al²⁴ stated that the leg volume continued to increase over a 2½ hour period during quiet standing, with filtration continuing indefinitely in the skin and gastrocnemius muscles which they considered to be low pressure filtration areas, and that it ceased rapidly in the other muscles of the leg which they considered high pressure areas.

Henry and Gauer²⁵ studying the effect of temperature on direct venous pressure in the normal foot found that as the temperatures increased, the mean venous pressure rose until, when the subject was hot (35 - 40° C.), it greatly exceeded the pressure opposing filtration.

Indeed, Landis et al²⁶ listed the factors affecting fluid passage through capillary walls as (1) permeability of capillary wall (2) capillary pressure (3) colloid osmotic pressure of the blood (4) temperature and (5) tissue pressure.

The rate of filtration decreased as tissue pressure rose in sudden obstruction and

Vascular Problems of the Lower Extremities

could in some instances be stopped entirely with resultant necrosis. With sustained venous obstruction McMaster²⁷ was able to demonstrate a rise of the interstitial pressure of the skin of the leg in man from a normal of 25. - 37. mm. water to 150. - 230. mm. water after 15-27 minutes. Some of his experimental subjects experienced a sensation of relief from congestion after about 20 minutes of sustained venous obstruction as if some tissue adjustment or some venous by-pass to the marrow had occurred preventing further rise in pressure.

PATHOLOGICAL CONSIDERATIONS

Any alteration of structure and/or function which impedes the normal return of blood and/or lymph from the peripheral circulation to the heart and specifically causes pathologic changes in the lower extremities would be pertinent to this discussion.

Foote et al²⁸ studying 200 unselected cases equally divided between the OPD and IPD at Massachusetts General Hospital to see what was the underlying cause of the bilateral edema of the lower extremities reported the following etiologies:

| Out-Patient Cases | Nos. | In-Patient Cases | Nos. |
|----------------------------------|------|--|------|
| Varicose veins | 56 | Congestive failure | 60 |
| (Varicose veins with obesity) | 31 | (Varicose veins with or without obesity or vice versa) | 12 |
| (Varicose veins without obesity) | 25 | Renal disease | 7 |
| Obesity alone | 13 | Nutritional | 7 |
| Cardiac failure alone | 13 | Hepatic cirrhosis | 7 |
| Lymphedema | 4 | Leukemia | 2 |
| Renal disease | 3 | Myxedema | 2 |
| Nutritional | 3 | Miscellaneous | 3 |
| Hepatic cirrhosis | 2 | | |
| Miscellaneous | 6 | | |

Mahorner²⁹, in 1949, reviewing indurated leg and leg ulcers defined indurated leg as "any hardness or thickening of the skin and subcutaneous tissue resulting from edema" and listed the factors causing indurated leg as follows:

- | | |
|------------------------------------|----------------------------------|
| 1. Post-thrombophlebitic edema | 6. Lymphedema |
| 2. Varicose veins | 7. Immersion leg |
| 3. Obesity | 8. Arterio-venous communications |
| 4. Trauma | 9. Systemic causes |
| 5. Infection (pyogenic or fungous) | 10. Combination of the above |

He noted that the most frequent cause was post-thrombophlebitic edema and he repeatedly emphasized the importance of preventing the recurrence of swelling in the leg day after day which he felt was the direct cause of indurated leg and its allied disorders. This symptom-complex: progressive edema, induration, ulceration and/or eczematization, when the end-result of thrombophlebitis, seems to be best summed up by considering these stages as all part of the post-thrombophlebitic syndrome.

Of even greater significance was his statement to the effect that, "It seems always fair and even important to tell the patient that whereas varicose veins can be improved by ligation, the tendency to develop varicose veins cannot thus be removed."

Hunter et al³⁰ studying 351 autopsies found thrombosis of the deep leg veins to be present in 52.7% of middle-aged to older patients who had to be in bed for

varying periods of time. The majority of the thromboses were in the larger veins and were more frequent in the soleus than the gastrocnemius with a slightly greater tendency to occur in the right leg. Thromboses were more frequently bilateral than unilateral in their series.

Gunnar Baur,¹ the moderator of his section on post-thrombophlebitic sequelae at the 1951 International Congress of Surgery quoted the following:

1. A review of 100 cases of phlegmasia alba dolens in 1942 at the Marie Stadt Hospital showed that after 10 to 15 years all of the patients had edema in the leg, that 9/10's of them had indurated skin changes and 4/5's had ulceration.
2. Zilliacus in 1948 reported pronounced and often severely disabling sequelae in 90% of 680 follow-up cases. In 510 of these patients he found that at least 81 had to change their occupation and 54 had become definitely unfit for all kinds of work.
3. Sturup reporting from Denmark in 1950 noted in his post-thrombophlebitis, there was a recurrence rate of ulceration at least once every second year, hospitalization every fourth year and that these people were unable to work for about 25 days out of each year.
4. Birger in the 1941 report based on figures from the Swedish Pensions Board noted that as a cause of incapacity, this affection came a bit ahead of such important disease groups as tuberculosis of joints or bones and diabetes mellitus.

In recent reports Moyer and Butcher¹¹ and Butcher and Hoover³¹ extended McMaster's work to demonstrate the following features about the superficial cutaneous lymphatics in addition to their being obliterated by sterile inflammation and showing regrowth following incisions.

The lymphatics were usually absent in skin about stasis ulcers and over "hard lymphedema" but were dilated over soft lymphedema. They were normal over simple varicosities and in cardiac and nutritional edemas. In split-thickness autografts they were at first dilated and if the graft was to survive, the lymphatics had to survive and establish connections with adjacent skin lymphatics. Following simple incision and closure, these lymphatics would by the 12-14th day post-operatively establish these connections if healing was by primary intention. In healing by secondary intention the lymphatics were fewer in number and smaller in caliber because of the scar.

The absence of superficial cutaneous lymphatics was associated with hyperkeratoses, collagenous and fibrous hyperplasia of the dermis, fibrous dysplasia of the subcutaneous fat and collagenous hyperplasia of the enveloping muscular fascia.

Despite all these findings there is a paucity of literature on values for the normal and abnormal lymphatic pressures in the lower extremities and the role of the deeper lymphatics in these problems other than in phlegmasia alba dolens.

For the various vein anomalies of the lower extremities and a discussion of the hereditary factors leading to venous difficulties the author refers the reader to any standard text on the subject.

Vascular Problems of the Lower Extremities

SUMMARY

The basic anatomical, physiological, and pathological considerations have been reviewed and their inter-relationship stressed. The relative inadequacies of our present-day information in some of these fundamental facets have been either pointed out or inferred in the hope that other investigators might see fit to fill these gaps.

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Beninson

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